

Amendments to the Claims:

This listing of claims will replace all prior versions and listings of claims in the application:

Listing of Claims:

Claims 1-4 (Cancelled)

Claim 5 (Withdrawn): The method of claim 1, wherein said inhibitor is a peptidomimetic or a polypeptide containing 2-10 aminoacids.

Claim 6 (Withdrawn): The method of claim 1, wherein said inhibitor is a small molecule with molecular weight less than 700 Daltons.

Claims 7-21 (Cancelled)

Claim 22 (Withdrawn): A method of inhibiting complement activation comprising the steps of:

(a) selecting an inhibitor molecule from the group consisting of peptidomimetic, peptides containing 2-20 aminoacids, oligonucleotides containing 2-20 nucleotides, and small organic molecule with molecular weight is less than 2000 Daltons;

(b) establishing by *in vitro* assay procedures that said inhibitor binds to factor B; and

(c) establishing by *in vitro* assay procedures that said inhibitor further prevents factor B binding to properdin and properdin-bound C3b; prevents the release of Bb; reduces C3a, C5a, and C5b-9 generation in blood; reduces C3 conversion into C3a and C3b; reduces C5 conversion into C5a and C5b; reduces the activation of neutrophils, monocytes and platelets; or inactivates the cells bearing C3a and C3a receptors.

Claim 23 (Withdrawn): The method of claim 22, wherein said inhibitor is a peptidomimetic or a polypeptide containing 2-10 aminoacids.

Claim 24 (Withdrawn): The method of claim 22, wherein said inhibitor is a small molecule with molecular weight less than 700 Daltons.

Claim 25 (Currently amended): A method of inhibiting factor B-dependent complement activation in blood of a subject in need thereof, comprising administering to blood of the subject an a therapeutically effective amount of an anti-factor B antibody effective to inhibit complement activation, the anti-factor B antibody in an *in vitro* assay having a greater effectiveness at preventing factor B binding to properdin-bound C3b than preventing factor B binding to and properdin-bound-free C3b; preventing the formation of Bb; reducing C3a, C5a, and C5b-9 generation; reducing C3 conversion into C3a and C3b; reducing C5 conversion into C5a and C5b; reducing the activation of neutrophils, monocytes and platelets; and inactivating

cells bearing C3a and C5a receptors, wherein administration of the therapeutically effective amount of the antibody does not decrease factor B levels in the blood.

Claim 26 (Previously Presented): The method of claim 25, wherein the antibody specifically binds to factor B and factor B fragments.

Claim 27 (Previously Presented): The method of claim 25, wherein the antibody or an antibody fragment thereof is monoclonal, polyclonal, recombinant, chimeric, humanized or human antibody.

Claim 28 (Currently Amended): The method of claim 25, wherein the antibody comprising a fragmented antibody selected from the group consisting of F(ab), F(ab'), F(ab)₂, ~~F(ab)₂~~ and scFv.

Claim 29 (Previously Presented): The method of claim 25 wherein the complement alternative pathway is substantially inhibited.

Claim 30 (Previously Presented): The method of claim 25, wherein the complement activation is associated with inflammation in a subject.

Claim 31 (Previously Presented): The method of claim 25, wherein the complement activation is associated with a disease.

Claim 32 (Previously Presented): The method of claim 31, wherein the disease comprises at least one of myocardial infarction, ischemia/reperfusion injury, stroke, acute respiratory distress syndrome (ARDS), burn injury, cardiopulmonary bypass inflammation, extracorporeal circulation, radiographic contrast media induced allergic response, transplant rejection, multiple sclerosis, myasthenia gravis, neuronal injury, pancreatitis, miscarriages, rheumatoid arthritis, Alzheimer's disease, asthma, thermal injury, anaphylactic shock, bowel inflammation, urticaria, angioedema, vasculitis, Sjogren's syndrome, lupus erythromatosus, and membranous nephritis, dermatomyositis, vascular stenosis or restenosis.

Claim 33 (Currently amended): A method of treating disease pathologies associated with complement activation mediated by factor B in human blood, the method comprising administering to human blood a therapeutically effective amount of an antibody that binds to factor B present in the human blood, the anti-factor B antibody in an in vitro assay having a greater effectiveness at preventing factor B binding to properdin-bound C3b than preventing factor B binding to and properdin-bound free C3b; preventing the formation of Bb; reducing C3a, C5a, and C5b-9 generation; reducing C3 conversion into C3a and C3b; reducing C5 conversion into C5a and C5b; reducing the activation of neutrophils, monocytes and platelets; and inactivating cells bearing C3a and C5a receptors, wherein administration of the therapeutically effective amount of the antibody does not decrease factor B levels in the blood.

Claim 34 (Previously Presented): The method of claim 33, wherein the antibody or fragment thereof is a monoclonal, polyclonal, recombinant, chimeric, humanized or human antibody.

Claim 35 (Currently amended): The method of claim 33, wherein the antibody comprising a fragmented antibody is selected from the group consisting of a F(ab), F(ab'), F(ab)2, F(ab)2₂, or scFv.

Claim 36 (Previously Presented): The method of claim 33, wherein the complement alternative pathway is substantially inhibited.

Claim 37 (Previously Presented): The method of claim 33, wherein the complement activation is associated with inflammation in a subject.

Claim 38 (Cancelled)

Claim 39 (Previously Presented): The method of claim 33, wherein the disease comprises at least one of myocardial infarction, ischemia/reperfusion injury, stroke, acute respiratory distress syndrome (ARDS), burn injury, cardiopulmonary bypass inflammation, extracorporeal circulation, radiographic contrast media induced allergic response, transplant rejection, multiple sclerosis, myasthenia gravis, neuronal injury, pancreatitis, miscarriages, rheumatoid arthritis, Alzheimer's disease, asthma, thermal injury, anaphylactic shock, bowel inflammation, urticaria,

angioedema, vasculitis, Sjogren's syndrome, lupus erythromatosus, and membranous nephritis, dermatomyositis, vascular stenosis or restenosis.

Claim 40 (Currently amended): A method of inhibiting complement alternative pathway activation associated with extracorporeal circulation inflammation, the method comprising;

contacting factor B in blood of a subject ~~going~~ undergoing extra corporeal circulation with a therapeutically effective amount of an antibody that specifically binds to factor B, the anti-factor B antibody in an *in vitro* assay having a greater effectiveness at preventing factor B binding to properdin-bound C3b than factor B binding to and properdin-bound free C3b, preventing the formation of Bb; reducing C3a, C5a, and C5b-9 generation in blood, reducing C3 conversion into C3a and C3b; reducing C5 conversion into C5a and C5b; reducing the activation of neutrophils, monocytes and platelets and inactivating cells bearing C3a and C5a receptors, wherein administration of the therapeutically effective amount of the antibody does not decrease factor B levels in the blood.

Claim 41 (Previously Presented): The method of claim 40, wherein the antibody or fragment thereof is a monoclonal, polyclonal, recombinant, chimeric, humanized or human antibody.

Claim 42 (Currently amended): The method of claim 40, wherein the antibody comprises a fragmented antibody selected from the group consisting of F(ab), F(ab'), F(ab)2, ~~F(ab)2~~, or scFv, 11.

Claim 43 (Previously Presented): The method of claim 40, wherein the complement alternative pathway is substantially inhibited.

Claim 44 (Previously Presented): The method of claim 40, wherein complement activation is associated with inflammation in a subject.

Claim 45 (Cancelled)